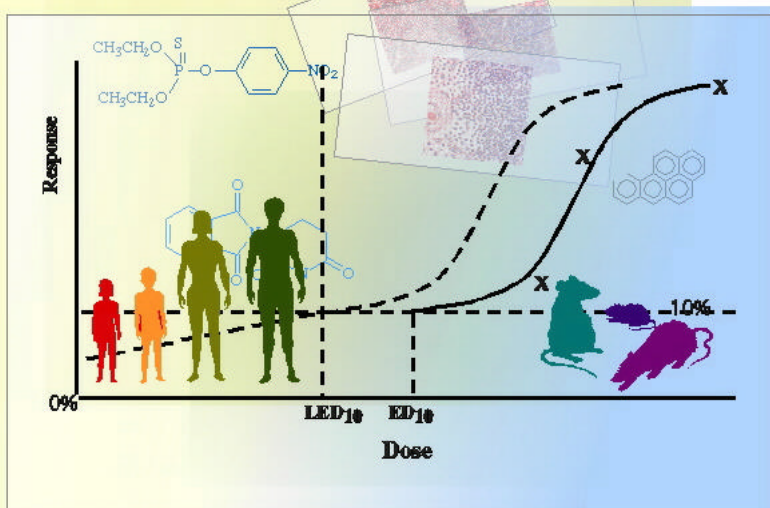


HUMAN HEALTH RISK ASSESSMENT

Methyl Parathion



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Protection Agency
Office of Pesticide Programs
Health Effects Division (7509C)

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August 2, 1999

HUMAN HEALTH RISK ASSESSMENT

Methyl Parathion

Phase 4

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METHYL PARATHION

Executive Summary

Uses

Methyl parathion, O, O-Dimethyl O-(4-nitrophenyl) phosphorothioate, is an acaricide and an insecticide registered for use on a variety of food and feed crops. Methyl parathion is a restricted use pesticide that is formulated as a microencapsulate [20.9% active ingredient (ai)], and an emulsifiable concentrate (ranges from 11.2 to 54.8% ai). Currently, a granular formulation is available but is not being supported for reregistration and is not included in the risk assessment. Methyl parathion is sold in the U.S. by Cheminova Agro A/S and Griffin Corporation, the basic producers, under the trade name Methyl Parathion, and by Elf Atochem North America, a formulator for Penncap-M®. Methyl parathion can be applied with aerial equipment, and an airblast sprayer (microencapsulated formulation only), by chemigation (microencapsulated formulation only), and with groundboom equipment. Both the registrant's proposed maximum application rates and the current label maximum application rates were used in this assessment. These application rates vary from 0.25 to 3.0 lbs. ai/A. Methyl parathion is formulated with several other active ingredients including ethyl parathion, malathion, and endosulfan.

Methyl parathion is a restricted-use pesticide and is available for retail sale to, and for use by, certified applicators (or persons under their direct supervision), and only for those uses covered by the certified applicator's certification. There are no labeled uses for homeowners. However, residential exposure could occur via agricultural spray drift from the use of methyl parathion on fields adjacent to residences or from the use of methyl parathion as a mosquito control agent. A mosquito control use (larvicide) is not being supported for reregistration by the primary data-submitter, Cheminova. The Agency contacted Health and Human Services and determined that methyl parathion has not been for many years, nor is, in use for mosquito control. A quantitative exposure and risk assessment for residential exposure via agricultural spray drift has not been completed as part of this risk assessment as the methodology for this assessment is still under development by the Agency.

Endpoints

The toxicity endpoints selected for the risk assessment are based primarily on neurotoxic effects, including neuropathology and cholinesterase (ChE) inhibition in the brain, red blood cell (RBC), and plasma, as well as behavioral effects and systemic toxicity (decreased hematocrit and erythrocyte levels). In addition, a single oral exposure to methyl parathion (7.5 mg/kg) in rodents resulted in peripheral nerve demyelination (tibial and sural nerves, dorsal and ventral root fibers). Additional effects of chronic exposure include retinal degeneration and sciatic nerve degeneration. No evidence of carcinogenicity was seen in any study.

An uncertainty factor (UF) of 100 was applied to the doses selected for risk assessment to account for both interspecies extrapolation and intraspecies variability. An additional factor of 10X was retained in accordance with the FQPA for the dietary risk assessment. In accordance with current HED guidance, the FQPA factor is not retained for the occupational risk assessment.

The Agency conducted the human health risk assessment for all registered uses of methyl parathion which were being supported under reregistration, plus hops, as well as for the use changes which reflect mitigation measures.

Dietary Assessment

Current tolerances for methyl parathion are based on the parent compound alone. This is consistent with Codex. The methyl parathion residues of concern that are included in this dietary risk assessment, based on ChE inhibition, are methyl parathion and its oxygen analog, methyl paraoxon. A dietary exposure assessment for methyl parathion residues from animal commodities was not performed since there are no tolerances currently established for residues of methyl parathion in meat, milk, poultry, and eggs. Residues of methyl parathion were not detected in ruminant tissue, milk, and egg samples collected from the ruminant and poultry metabolism studies. The pre-mitigation dietary exposure assessment was limited to those agricultural uses of methyl parathion which were being supported under reregistration. The United States Department of Agriculture (USDA) was contacted and any agricultural uses that USDA - IR4 wished to retain were included in the assessment. It was determined that hops were the only IR4 crop of agricultural interest that Cheminova would not support. Therefore, hops were included in the assessment. Dietary exposure estimates were refined to include monitoring data, percent crop treated data, and available processing and cooking data.

The acute dietary risk assessment (probabilistic), based on USDA's Continuing Survey of Food Intake by Individuals (CSFII) food consumption survey and using an acute Population Adjusted Dose (aPAD) of 0.00011 mg/kg/d, shows that acute dietary exposure to all population subgroups prior to mitigation measures exceeded the aPAD at the 99.9th percentile estimated exposure level (U.S. population 378% aPAD, children 1-6 years 881% aPAD). Children 1-6 years were identified as the most highly exposed population subgroup. Acute dietary exposure to children 1-6 years exceeded the aPAD at the 99th and 95th percentile also. Dietary exposure to children 1-6 years did not exceed the aPAD at the 90th percentile. Dietary risk estimates that reflect mitigation measures show that exposures from food do not exceed the aPAD for any population subgroup at the 99.9th percentile (U.S. population 60% aPAD, children 1-6 years 78% aPAD). Several crops were identified as making substantial contributions to the dietary risk. In other words, residues measured on these crops and the surveyed consumption of these crops by the different population subgroups, factored together, results in these crops making a large contribution to the overall estimated exposure. For methyl parathion, the largest contributors to the acute dietary exposure were identified as apples, peaches, grapes, and pears. It should be noted that only the use of the

microencapsulate (Mcap) formulation of methyl parathion on pome and stone fruits (apples, peaches, pears, and grapes) was being supported under reregistration, not the emulsifiable concentrate (EC).

Based on a refined pre-mitigation chronic dietary exposure analysis and using a chronic PAD (cPAD) of 0.00002 mg/kg/d, chronic dietary exposure to all population subgroups did not exceed the cPAD (U.S. population 17% cPAD, children 1-6 years 47% cPAD). Dietary risk estimates that reflect mitigation measures further reduced exposures from food and do not exceed the cPAD for any population subgroup (U.S. population 3% aPAD, children 1-6 years 8% aPAD)

Drinking Water Assessment

Ready to drink, treated drinking water data for methyl parathion, or “at the tap” water data, are not available. While the Agency’s Office of Water (OW) has established a lifetime health advisory (HA) of 2 ppb, methyl parathion does not have an established Maximum Contaminant Level, and it is not included on the OW’s Unregulated Contaminant Monitoring List. Therefore, public drinking water supply systems are not required to analyze for methyl parathion. Consequently, EFED relied on simulation models and some very limited surface-water monitoring data for this risk assessment. The monitoring data represent only a very small range of conditions (regional weather, streamflow, application rates and methods) and it cannot be assumed that they represent surface water concentrations or conditions across the United States. None of the monitoring data included analysis for the methyl paraoxon metabolite. Although the Agency considers it unlikely that drinking water concentrations “at the tap,” will make the largest, or a significant, contribution to the total dietary burden, there is sufficient information from the available monitoring data and the models to warrant close monitoring of potential surface and ground water sources of methyl parathion exposure.

Occupational Assessment

HED has determined that there are potential occupational exposures of concern to mixers, loaders, applicators, and other handlers associated with uses of methyl parathion. Calculations of occupational risk were based on combined dermal and inhalation exposures, a No Observable Adverse Effect Level (NOAEL) = 0.11 mg/kg/day, and 100% dermal and inhalation absorption. The risk calculations indicate that the Margins of Exposure (MOE) less than 100 with maximum risk reduction measures for nearly all of the supported short- and intermediate-term occupational exposure scenarios (many less than 1). Depending on crop and postapplication activities, calculated re-entry intervals (REI) for workers, that would not be of concern, were estimated to range from 30 to 33 days for Mcap formulations, and from 7 to 9 days for EC formulations. Current labels show 48-72 hours REI. Mitigation measures have

eliminated many of the activities involving hand harvesting and therefore, have reduced occupational risks. Risk estimates of residential dermal and inhalation exposures were not estimated. The Agency is currently developing methods to assess residential risks, and these risks will be assessed in the future when these new methods are available. However, based on available information, HED remains concerned about residential risks from methyl parathion spray drift.

Aggregate Assessment

Under the Food Quality Protection Act, the Agency considers contributions to risk from various exposure sources, specifically, food, drinking water, and residential. Methyl parathion has no registered residential uses, therefore only exposures through food and drinking water were considered in the aggregate risk assessment. The acute aggregate risk estimate for all registered uses, pre-mitigation, indicated that there is no room for exposure to methyl parathion in drinking water because risk from food sources alone exceed the Agency's level of concern ($> 100\%$ aPAD). The acute aggregate risk estimate which reflects mitigation measures may still be of concern. Though acute exposure to methyl parathion from food sources alone, with mitigation measures, does not exceed the Agency's level of concern ($< 100\%$ aPAD), limited surface water monitoring data indicate potential exposures at unacceptable levels. However, without actual drinking water monitoring data, it is difficult to draw any conclusions about actual residues in drinking water. The chronic aggregate risk assessment is not of concern, pre- or post-mitigation measures.

The Agency is in the process of formulating guidance for conducting cumulative risk assessment. When the guidance is finalized, methyl parathion and other organophosphates will be revisited to assess the cumulative effects of exposure to multiple organophosphates.

Reported Incidents

A review of the published incident data indicates that in outdoor agricultural uses, the primary activities associated with poisoning are application and spray drift (Attachment 11). Methyl parathion is associated with less poisoning compared to other organophosphate or carbamate pesticides when adjusted for the number of incidents per amount of use (lbs ai/A).

I. Hazard Characterization

A. Hazard Profile

The toxicological database is complete pending submission of a developmental neurotoxicity study. In summary, methyl parathion is acutely toxic (category 1) for oral, dermal, and inhalation routes, is slightly-moderately irritating to the eyes and skin, and is not a dermal sensitizer. The toxicity endpoints selected for the risk assessment are based primarily on neurotoxic effects, including neuropathology and ChE inhibition in the brain, RBC, and plasma, as well as behavioral effects and systemic toxicity. A single exposure to methyl parathion (7.5 mg/kg) in rodents results in peripheral nerve demyelination (tibial and sural nerves, dorsal and ventral root fibers). Chronic exposure at a dose level of 2.21 mg/kg/d results in retinal degeneration and sciatic nerve degeneration. There are no notable differences in sensitivity to methyl parathion between male and female animals. No evidence of carcinogenicity was seen in any study. Methyl parathion is classified as a "Group E" carcinogen, indicating no evidence of carcinogenicity in humans; i.e., the chemical is characterized as "Not Likely" to be carcinogenic in humans via relevant routes of exposure. This classification is supported by the lack of mutagenic activity. There is evidence suggesting that methyl parathion may function as an endocrine disruptor (Attachment 3).

Table 1. summarizes the acute toxicity data for the technical methyl parathion.

Table 1. Acute Toxicity Data for Methyl Parathion (Technical)

Guideline No.	Study Type	MRID #	Results	Toxicity Category
81-1	Acute Oral (rat)		LD ₅₀ = 4.5-24 mg/kg	I
81-2	Acute Dermal (rat)		LD ₅₀ = 6 mg/kg	I
81-3	Acute Inhalation (rat)	256961	LC ₅₀ < 0.163 mg/L (< 7 mg/kg)	I
81-4	Primary Eye Irritation	256966, 40542602	Irritation clear by 7 days	III
81-5	Primary Skin Irritation	256962	Max. score = 2.0; 72 h = 0.5	IV
81-6	Dermal Sensitization	256963	Negative	
81-8	Acute Neurotoxicity Delayed Hen	41606801	Negative	

No dermal absorption study was available. Although there was a 21-day dermal toxicity study in rabbits available, it was not selected to generate a dermal toxicity endpoint for the following reasons: 1) The rabbit is less sensitive than the rat to this chemical (for example, in the rabbit developmental study, the 3.0 mg/kg/d dose resulted in only minimally significant ChE inhibition, and in the rat, maternal deaths occurred in the developmental toxicity study at the same dose), 2) several endpoints (including neurotoxicity and neuropathology) occurring at low doses in the acute oral rat study were not measured in the dermal rabbit study, 3) oral and dermal effects seen in other acute studies occurred at similar doses (Attachments 1 and 2), so there is no reason to believe that neurotoxic effects might not occur at low dermal doses, and 4) because of physiological and biochemical factors, unique to the rabbit, which might result in an underestimation of the dermal toxicity of organophosphorus pesticides belonging to the thiophosphate subgroup (R. Zendzian, HED, memo dated March 1997). Therefore, based on available information, including comparison of toxicity following oral and dermal exposure, dermal absorption was estimated to be 100% (i.e. equivalent toxicity is expected after oral or dermal exposure to a given amount of methyl parathion). This decision was reevaluated and reaffirmed in the Hazard Identification Assessment Review Committee (HIARC) meeting of March 4, 1999 (Attachment 2). Cheminova submitted a 5-day dermal toxicity study in rats (06/03/99) and it is in review.

B. Endpoint Selection

Previously, the HIARC selected a NOAEL = 0.025 mg/kg/d from an acute neurotoxicity study for use in acute dietary and short-term occupational risk assessment (Attachment 1). The dose spacing in Cheminova's submitted acute neurotoxicity study was very broad and the NOAEL of 0.025 mg/kg/d was believed to possibly be an artifact of the doses selected for the study (LOAEL = 7.5 mg/kg/d). Following a review of the comments submitted by Cheminova in Phase 3 of the Public Participation Process, the HIARC reevaluated the endpoints on March 4, 1999, and determined that the acute dietary, as well as the dermal and inhalation short- and intermediate-term occupational endpoints should be based on a NOAEL of 0.11 mg/kg/d for inhibition of plasma, brain, and RBC ChE and neuropathology seen in a 1 year dietary study in rats at the LOAEL of 0.53 mg/kg/d (Attachment 2). The NOAEL of 0.11 mg/kg/d is still considerably lower than the LOAEL from the previously selected acute neurotoxicity study (7.5 mg/kg/d).

Cheminova submitted an acute dietary risk assessment (conducted by Novigen Sciences, Inc.) on 3/16/99. The acute dietary endpoint used was based on a NOAEL = 1 mg/kg/day for inhibition of RBC ChE at 1.5 mg/kg/day (the LOAEL) in Cheminova's newly conducted acute feeding study in the rat.

This acute feeding study has recently been submitted (05/99) to the Agency and is in review. Cheminova's acute feeding study was conducted using a novel protocol, not previously submitted to the Agency (not guideline), and will undergo peer review (Science Advisory Panel) following internal review.

The HIARC did consider the registrant's proposals for the other endpoints but reaffirmed that the NOAEL = 0.02 mg/kg/d from the 2-year chronic oral study in the rat should be used for the chronic dietary risk assessment. The HIARC also reaffirmed that the dermal absorption factor for methyl parathion would continue to be 100% for risk assessment purposes (Attachment 2). Due to the high toxicity seen in the submitted acute inhalation study, 100% absorption is considered appropriate. Details of the HIARC's findings and rationale can be found in the attached Revised Toxicology chapter (Attachment 3), the addendum to the HIARC memo (Attachment 2), and the original HIARC endpoint selection document (Attachment 1).

Table 2. Revised Methyl Parathion Endpoints (3/4/99)

Exposure Duration	Exposure Route	Endpoint		Comments
		Dose	Effect	
Acute - PAD	Dietary	aPAD = 0.00011 mg/kg/d	Neuropathology and inhibition of brain, plasma, and RBC ChE	NOAEL = 0.11 mg/kg/d. Based on neurotoxicity, neuropathology and inhibition of brain, plasma, and RBC ChE occurring at 0.53 mg/kg/d. One year dietary study in rats. UF of 100 applied for intra and inter species differences and an additional safety factor of 10X retained by the FQPA Safety Factor Committee for FQPA.
Chronic - PAD	Dietary	cPAD = 0.00002 mg/kg/d	Systemic toxicity, neuropathology, and inhibition of RBC ChE at the LOAEL	NOAEL = 0.02 mg/kg/d. Based on systemic toxicity, neuropathology, and RBC ChE inhibition occurring at 0.21 mg/kg/d. Inhibition of plasma and brain ChE occurred at higher doses. Retinal degeneration and clinical signs occurred at the highest dose. 2-Yr chronic feeding study in rats. UF of 100 applied for intra and inter species differences and an additional safety factor of 10X retained by the FQPA Safety Factor Committee for FQPA.
Short-term (1-7 days) Occupational	Dermal	NOAEL = 0.11 mg/kg/d	Neuropathology and inhibition of brain, plasma, and RBC ChE	Same endpoint as aPAD. Although a 21-day dermal study in the rabbit is available, it was not selected. See Hazard ID SARC memo 12/01/97. Dermal absorption rate estimated to be 100% (Revisited 02/14/99, 03/04/99). UF of 100 applied for intra and inter species differences.
Intermediate- term (7 - 90 days) Occupational	Dermal	NOAEL = 0.11 mg/kg/d	Neuropathology and inhibition of brain, plasma, and RBC ChE	Same endpoint as aPAD. Long term dermal study not available. Dermal absorption rate estimated to be 100%. UF of 100 applied for intra and inter species differences.
Short- & Intermediate-term Occupational	Inhalation	NOAEL = 0.11 mg/kg/d	Neuropathology & inhibition of brain, plasma, and RBC ChE	Same endpoint as aPAD. Due to high toxicity seen in acute inhalation study, 100% absorption is estimated. UF of 100 applied for intra and inter species differences.

C. FQPA Considerations

The decision to retain the full 10X FQPA Safety Factor was based on a substantial data gap that can be filled with the submission of a Developmental Neurotoxicity Test. The data that were instrumental in this decision are discussed below.

Neuropathology reported in acceptable studies submitted by the registrant:

- ☐ Neuropathology seen in experimental animals in the guideline acute neurotoxicity study;
- ☐ Neuropathology seen in experimental animals in the guideline chronic/carcinogenicity study;
- ☐ Neuropathology seen in experimental animals in the non-guideline, but acceptable one year neurotoxicity study.

Fetal/neonate susceptibility reported in open literature citations which were retrieved and reviewed by the Agency:

- ☐ An open literature citation which assessed postnatal functional toxicity following prenatal exposure reported the inhibition of acetyl cholinesterase and other neurochemical biomarkers in pups which persisted to day 28 and impaired behavioral parameters (Gupta *et. al.* 1985);
- ☐ Additional open literature citations reported that neonates were more sensitive to acute lethality from methyl parathion than adults and that significant compound-related and age-related differences in duration of ChE inhibition can occur (Pope *et al.* 1991, Pope and Chakraborti 1992);
- ☐ Possible endocrine disruption in mammals (Dhondup and Basavanneppa 1997, Lukaszewica-Hussain, Moniuszko-Jakoniuk and Pawlowska 1985).

Fetal/neonate sensitivity/susceptibility reported in studies submitted by the registrant during the comment period:

- ☐ Decreased survival and convulsions in the surviving F_{1b} pups were reported in a non-guideline multi-generation reproduction study in rats;
- ☐ Embryotoxicity or fetotoxicity was observed at non-maternally toxic levels

in an additional supplementary developmental study in rats which had previously been submitted to the Agency.

The standard guideline studies for developmental and reproductive toxicity, which have been submitted by the registrant and are acceptable, are not required to measure cholinesterase inhibition, behavioral effects, neuropathology, or increased sensitivity to lethal effects in pups. Thus, these studies are silent on effects that have been reported in the open literature. Even though the open literature studies have a number of deficiencies, the fact that *several* studies have reported adverse effects on neonates raises concern. The suggestive evidence of possible endocrine disruption, although not heavily weighted, was also taken into account. If the information from these studies is considered together with the reported neuropathology seen in adult animals after a single and multiple doses of methyl parathion and the results from the supplementary developmental and reproduction studies submitted by the registrant which demonstrate fetal and neonate sensitivity, the concern for effects on the developing organism increases. Thus all of these data, taken *in toto* require that the 10X FQPA Safety Factor be retained until such time as the Agency receives an acceptable Developmental Neurotoxicity Test. When this study is received and reviewed, the final decision on the retention, reduction, or removal of the 10X FQPA Safety Factor will be made based upon the weight of the evidence.

II. Exposure Characterization

A. Registered Uses

Methyl parathion is registered for use on a variety of fruits, vegetables, and feed crops. Methyl parathion is sold in the U.S. by Cheminova Agro A/S and Griffin, the basic producers, under the trade name Methyl Parathion, and by Elf Atochem North America, a formulator for PennCap-M®. Registered formulations for use on food and feed crops include Mcap and EC formulations. Currently, a granular formulation is available but is not being supported for reregistration. Methyl parathion can be applied with aerial equipment and an airblast sprayer (Mcap formulation only), by chemigation (Mcap formulation only), and with groundboom equipment. Methyl parathion is formulated with several other active ingredients including ethyl parathion, malathion, and endosulfan.

The following uses (1-4) are currently being supported by the registrant and are included in this assessment:

1. Food, Forage, Feed, and Fiber Crops

Alfalfa, artichoke, barley, beans, broccoli, Brussels sprouts, cabbage, canola, carrot, cauliflower, celery, collards, corn, cotton, grass forage/fodder/hay, hops, kale, lentils, lettuce, mustard greens, oats, onion, pastures, peas, potato, rangeland, rice, rye, soybeans, spinach, sugar beet, sunflower, sweet potato, tomato, turnip, wheat, and yam.

2. Fruits and Nuts

Almond, walnut, peanut, pecan, apple, cherry, grapes, nectarine, peach, pear, and plum.

3. Ornamental Plants and Forest Trees

Christmas tree plantations, forest trees, ornamental and/or shade trees, pine trees, field-grown ornamental herbaceous plants, and field-grown ornamental woody shrubs and vines.

4. Non-agriculture Land and Pastures

Rights-of-way and grazing lands.

The crops included in the post mitigation uses of methyl parathion differ from the above list. The following crops were added: dried beans and dried peas. The following crops were taken out: apple, artichoke, broccoli, Brussels sprouts, carrots, cauliflower, celery, cherry, collards, forest trees, garden beets, grapes, grasses grown for seed, kale, kohlrabi, lettuce, mustard, nectarine, non-agricultural land (mosquito use), ornamentals, pastures, peach, pears, plums, prunes, rangeland, spinach, succulent beans, succulent peas, tomatoes, and turnips.

B. Dietary Exposure

1. Food Exposure

The HED Metabolism Assessment Review Committee (Attachment 5) tentatively concluded that methyl parathion residues of concern in plant commodities include methyl parathion, methyl paraoxon, and *p*-nitrophenol, and that methyl parathion residues of concern in animal commodities include methyl parathion, methyl paraoxon, *p*-nitrophenol, and amino-paraoxon-methyl. The tolerance expression for plant and animal commodities is based on the parent methyl parathion only (U.S. tolerance definition is compatible with Codex). The methyl parathion residues of concern for plant and animal commodities included in this risk

assessment are based on ChE inhibition, and are methyl parathion and methyl paraoxon. Residues of *p*-nitrophenol are not included in the tolerance expression, nor considered in the aggregate risk assessment for methyl parathion with respect to cholinesterase inhibition, but should be considered in conjunction with the cumulative risk assessment for *p*-nitrophenol in the future. There is concern for the amino-paraoxon-methyl metabolite due to neuropathy of unknown etiology. Once outstanding livestock feeding studies have been submitted, the Agency will determine whether to include amino-paraoxon-methyl metabolite in the risk assessment.

Tolerances for residues of methyl parathion have been established on a variety of fruit, vegetable, and field crops. Additional magnitude of the residue and processing data remain outstanding. Anticipated residue (AR) estimates of methyl parathion and methyl paraoxon in/on plant commodities and processed commodities have been included in the dietary risk assessment for methyl parathion. Anticipated residue estimates are highly refined and are based on available monitoring and magnitude of the residue data. These estimates have been refined to include concentration/reduction factors determined from available processing data along with percent crop treated information.

No tolerances for residues of methyl parathion have been established in animal commodities (meat, milk, poultry, and eggs); although, tolerances for residues of methyl parathion have been established on numerous animal feed items. Therefore, the dietary exposure assessment may possibly underestimate dietary risks. Residues of methyl parathion were not detected in ruminant tissue, milk, and egg samples collected from the ruminant and poultry metabolism studies. Residues of methyl paraoxon were also not detected in any of the samples. Residues of methyl parathion were not detected in USDA monitored samples (1304 samples) of milk (1996-1998). Residues of methyl parathion detected in poultry tissue samples collected from the poultry metabolism study were very low. Based on available data, it is uncertain if finite residues of methyl parathion and methyl paraoxon are likely to occur in animal commodities; hence, AR estimates for residues of methyl parathion and methyl paraoxon in animal commodities were not included in the dietary risk assessment for methyl parathion. If required, appropriate tolerances for methyl parathion residues in animal commodities will be determined once data are available from outstanding livestock feeding studies.

2. Drinking Water Exposure

When the preliminary HED chapter (09/01/98) was written, potential exposure and risk from methyl parathion in drinking water were assessed using modeled estimates, and limited monitoring data. EFED provided HED with a Tier 2 surface water exposure assessment derived from the PRZMS3 model, which simulates the erosion and runoff from an agricultural field, and the EXAMS model, which simulates fate in a surface water body. A Tier 1 ground water exposure assessment was derived from the SCI-GROW screening model only, with no refinements. No further refinements can be made by EFED without ground water monitoring data.

Ready to drink, treated drinking water data for methyl parathion, or “at the tap” water data, are not available. While the Agency’s OW has established a lifetime HA of 2 ppb, methyl parathion does not have an established Maximum Contaminant Level, and is not included on the OW’s Unregulated Contaminant Monitoring List. Therefore, public drinking water supply systems are not required to analyze for methyl parathion. Consequently, EFED has relied on simulation models and other surface- and ground-water monitoring data for this revised risk assessment.

a. Surface Water

The surface-water concentrations estimated from the PRZM-EXAMS screening model (Tier 2) for human health risk assessments are: acute- 254 ppb ($\mu\text{g/L}$) and chronic- 4.2 ppb. However, these screening estimates are significantly higher than the concentrations seen in monitoring studies that have been obtained by EFED since the Preliminary HED chapter was issued (09/01/98). Data from targeted monitoring studies such as those in California, and the Mississippi River basin may provide a better estimate of possible acute drinking water concentrations than the models. In addition, Cheminova supplied supplementary information during Phase 3 of the Public Participation Process and suggested alternative input parameters for the modeled estimates.

Prior to a mitigation program instituted by California EPA's Department of Pesticide Regulation (CDPR) in the early 1990's, peak concentrations of methyl parathion in the Colusa Basin Drain were measured as high as 6 ppb. Although monitoring data are more realistic than modeling results, they do not necessarily reflect the use scenarios most vulnerable to contamination. For instance, the CDPR monitoring of the Colusa Basin Drain targeted methyl parathion use on rice. Application rates and the number of applications for many crops are higher than those for rice. In addition, current mitigation measures incorporating retention of water on treated fields is relevant only to rice, and not other crops to which methyl parathion is applied. Mitigation measures such as holding ponds lower the expected surface water concentrations, but to what extent is unknown, and it is not applicable to other crops.

Since EFED's preliminary chapter was issued, EFED has obtained targeted surface-water monitoring data collected by the United States Geological Survey (USGS) from rivers in the Mississippi Embayment cotton-growing region. Samples were drawn from five rivers in 1996 and 1997, and methyl parathion was detected in all five. Detected concentrations reached up to 0.42 ppb. The site with the highest frequency of detections in this study had 8 detections in 17 samples during water year (WY) 1996, and 8 detections in 37 samples during WY1997. However, the rivers sampled are not known drinking-water sources. Mississippi derives its drinking water almost exclusively from ground water, and of the five stations sampled for methyl parathion, only one was within 25 miles of a surface-water body used for drinking water. Usage data provided by Cheminova indicates that the Mississippi

Embayment cotton-growing region represents the area with the greatest density of methyl parathion use in the country.

In another 1996 monitoring program in the Mississippi Embayment region, the USGS detected methyl parathion in 18% of the 60 samples it collected from tributaries of the Mississippi River. The highest concentration detected was about 0.12 ppb, and the 50th percentile concentration was about 0.05 ppb.

The maximum acute surface water concentration simulated by PRZM/EXAMS was 214 ppb, for use on cotton at the maximum label rates. Cotton was chosen since it also has the highest application rate of all the use sites. When the input parameters, suggested by Cheminova in Phase 3 of the Public Participation Process, were considered in a hypothetical scenario, the peak concentration estimated for cotton was 17.8 ppb. Though somewhat refined, this is still considered a conservative estimate. Given the fact that the 0.42 and 6 ppb detections came from very limited, targeted surface-water monitoring studies on cotton and rice, respectively, and that the data represent only a very small range of conditions (a year or two of weather, streamflow, application rates and methods), insufficient evidence exists to determine how nationally representative these exposure concentrations are.

EFED has obtained some closer-to-the-tap targeted chronic monitoring data from Jefferson Parish, Louisiana, drawn at two intakes of a surface water derived drinking water plant on the Mississippi River. Weekly composites (continuous slow sampling to a refrigerated container over one week) were drawn for 52 weeks of the year. In 1994, raw water drawn at one intake had 18 detections of methyl parathion out of the 52 composite samples. At another intake in the same year, there were 21 out of 52 detections. The average for both plants was 0.009 ppb (detection limit) with highs of 0.03 and 0.04 ppb, respectively.

b. Ground Water

Using the screening model SCI-GROW, EFED calculated a ground water concentration of 0.6 ppb (Tier 1) for human health risk assessment. Data collected from a variety of sources did not identify any known instance in which a ground-water concentration higher than 0.6 ppb was detected, although individual detections have been within the same order of magnitude. Therefore, EFED suggests that 0.6 ppb is a reasonable conservative modeled estimate of possible acute concentrations of methyl parathion in drinking water derived from ground water. EFED does not have a model for estimating Tier 2 ground water concentrations for dietary

risk assessments.

C. Non-Dietary Exposure

1. Occupational Handler Exposure

HED has determined that there are potential short- and intermediate-term exposures to mixers, loaders, applicators, and other handlers during the usual use-patterns associated with methyl parathion. Based on the use patterns of methyl parathion, twelve major exposure scenarios were identified: (1a) mixing/loading liquids (EC) for aerial application; (1b) mixing/loading liquids (EC) for groundboom application; (2a) mixing/loading liquids (Mcap) for aerial/chemigation application; (2b) mixing/loading liquids (Mcap) for groundboom application; (2c) mixing/loading liquids (Mcap) for airblast application; (3) applying sprays with aerial equipment (EC); (4) applying sprays with aerial equipment (Mcap); (5) applying sprays with groundboom equipment (EC); (6) applying sprays with groundboom equipment (Mcap); (7) applying sprays with airblast sprayer (Mcap); (8) flagging sprays (EC); and (9) flagging sprays (Mcap).

Chemical-specific data for assessing human exposures during pesticide handling activities were not submitted to the Agency in support of the reregistration of methyl parathion. It is the policy of the HED to use data from the Pesticide Handlers Exposure Database (PHED) Version 1.1 to assess handler exposures for regulatory actions when chemical-specific monitoring data are not available. While data from PHED provide the best available information on handler exposures, it should be noted that some aspects of the included studies (e.g., duration, acres treated, pounds of active ingredient handled) may not accurately represent labeled uses in all cases. HED has developed a series of tables of standard unit exposure values for many occupational scenarios that are utilized to ensure consistency in exposure assessments. See Tables 2-6 in the revised Occupational and Residential Exposure and Risk Assessment Chapter (Attachment 14).

2. Postapplication Occupational Exposure

Chemical-specific postapplication exposure and/or environmental fate data have not been submitted by the registrants in support of reregistration of all formulation types of methyl parathion. In lieu of these data, a potential range of postapplication exposures were estimated to determine potential risks for the representative crops used in the handler exposure assessment section.

The surrogate assessment on pre mitigation uses for the Mcap formulation uses a typical transfer coefficient (T_c) for tree crops (peaches, apples and pears) of 10,000 cm^2/hr (based on HED's Exposure Science Assessment Committee Policy No. 3, "Agricultural Default Transfer Coefficients," May 7, 1998), from activities such as harvesting and pruning, and a typical T_c for grapes of 15,000 cm^2/hr , from activities such as harvesting and hand girdling. The dislodgeable foliar residue (DFR) is derived from the various application rates using an estimated 20% of the rate applied as initial dislodgeable residues, and an estimated 25% dissipation rate per day. The dissipation half-life of the Mcap formulation is 1 to 2 days (based on environmental fate data supplied by EFED). A half-life of 1 to 2 days has also been suggested for methyl paraoxon, depending upon the crop and climate. The estimated dissipation rate of 25% per day is intended to approximate this half-life. For grapes, the registrant's proposed application rate is 1.5 lbs ai/A and the current maximum label rate is 3.0 lbs ai/A. For apples, pears, and peaches the application rate is 2.0 lbs ai/A.

The surrogate assessment on post-mitigation uses for the Mcap formulation uses a typical transfer coefficient (T_c) for nut crops (almonds, walnuts, and pecans) of 10,000 cm^2/hr (based on HED's Exposure Science Assessment Committee Policy No. 3, "Agricultural Default Transfer Coefficients," May 7, 1998), from activities such as harvesting and pruning, and a typical T_c for grapes of 15,000 cm^2/hr , from activities such as shaking, raking, pole and picking up. The dislodgeable foliar residue (DFR) is derived from the various application rates using an estimated 20% of the rate applied as initial dislodgeable residues, and an estimated 25% dissipation rate per day. The dissipation half-life of the Mcap formulation is 1 to 2 days (based on environmental fate data supplied by EFED). A half-life of 1 to 2 days has also been suggested for methyl paraoxon, depending upon the crop and climate. The estimated dissipation rate of 25% per day is intended to approximate this half-life. For nut crops, the application rate is 2.0 lbs ai/A.

The post application assessment for the emulsifiable concentrate formulation is the same for the pre-mitigation uses and the post-mitigation uses. The surrogate assessment for the EC formulation uses a typical T_c for cotton of 1,000 cm^2/hr for scouting in the early season and 4,000 cm^2/hr for scouting in the late season. Since the dissipation rate is chemical specific, the DFR data were obtained from an open literature study done with methyl parathion. The DFR data were derived by combining the amount of methyl parathion with the amount of methyl paraoxon that were present on the foliage each day, after an initial

application of 1.0 lb ai/A. Since the maximum application rate for cotton is 3.0 lbs ai/A and is greater than 1.0 lb ai/A, the initial amount found on the leaf on day 0 was multiplied by the application rate of the crop. The data were log transferred and a regression analysis was done. The dissipation was determined from the regression data to be 63% per day. The predicted DFR from the regression analysis were then determined using this dissipation rate, starting at day 0 and then used to obtain the dose for each day.

3. Residential

Although methyl parathion is a restricted use pesticide that is only to be applied by certified applicators, HED believes that residential exposures may occur in several situations. First, residential exposures may occur from the use of methyl parathion as a mosquito control agent (as permitted on some current labels). Second, even though methyl parathion is a restricted use pesticide and some (but not all) labels state "Not for home use", the possibility exists for residential postapplication exposure from commercial application of methyl parathion to private orchards. Finally, residential exposures may result from spray drift from the aerial application of methyl parathion to agricultural fields adjacent to residential areas.

HED did not quantitatively assess the exposures and risks to individuals who live adjacent to farm fields and that could potentially be exposed to methyl parathion from spray drift. Methods to assess these risks are currently being developed by the Agency, and these assessments will be conducted in the future when these methods are available. However, based on current information, HED remains concerned about the potential risks from this source.

III. Risk Assessment/Characterization

Risk is a function of exposure multiplied by hazard (Risk = Exposure x Hazard). Exposure may be measured or modeled, depending on the available data. Ideally the exposure data would be chemical specific occupational or residential monitoring data, at the tap drinking water data, and close to the plate food residue data on all crops. In the absence of an ideal data set, surrogate data, and other factors are incorporated into the exposure assessments (dietary and non-dietary) to present a reasonable exposure picture based on the best available data. The hazard portion of the risk equation has several layers of safety built into it to provide a cushion between exposure and the dose at which adverse effects were seen in an animal study. Generally, endpoints are based on the dose at which **no** observable adverse effect is seen in an animal study. This is the No Observable Adverse Effect Level (NOAEL). The Lowest Observable Adverse Effect Level (LOAEL) is the next highest dose in an animal study, up from the NOAEL, at which the adverse effect of concern is seen. Levels of ChE inhibition which are of concern to the Agency do not always manifest themselves in clinical signs. In humans, the initial signs of organophosphate poisoning are headache, hypersecretion, muscle twitching, nausea, and diarrhea. Many of these symptoms are often confused with flu-like symptoms. Since the toxicity studies used for endpoint selection are conducted in animals, and there are differences between individual humans, additional uncertainty factors for inter- and intra-species variability are integrated into the hazard portion of the risk equation. Since the passage of the FQPA, an additional layer of protection is factored in (when appropriate) to provide an even greater safety cushion between exposure and toxic effects for particularly sensitive populations. It is in this light that expressions of risk (risk numbers) should be viewed with an understanding that they are not portrayals of imminent toxic effects to humans but as a measure of the distance between potential exposure and possible toxic effects.

In accordance with current HED policy (effective 03/11/99) the acute and chronic dietary endpoints are expressed as acute Population Adjusted Dose (aPAD) and chronic PAD (cPAD), and no longer as an adjusted Reference Dose (RfD).

$$\text{RfD} = \frac{\text{acute or chronic NOAEL}}{\text{Uncertainty Factor (UF)}}$$

Generally, an UF of 100 is applied for intra- and inter-species differences.

$$\text{PAD} = \frac{\text{acute or chronic RfD}}{\text{FQPA factor}}$$

The use of the PAD will apply whether the FQPA factor is retained (10x or 3x) or not (1x). When a PAD is used, such as in the dietary assessment, the risk is expressed as

a percentage of the PAD which is equal to the measured exposure divided by the PAD and then multiplied by 100 or:

$$\text{Risk (\% PAD)} = \frac{\text{Exposure}}{\text{PAD}} \times 100$$

Occupational, residential (when applicable), and the aggregate risk (when appropriate) will still be expressed as the Margin of Exposure (MOE).

$$\text{MOE} = \frac{\text{NOAEL}}{\text{Exposure}}$$

Current HED policy requires that FQPA safety factors be retained for dietary and non-occupational exposures, when appropriate, not occupational exposures. Therefore, an MOE of ≥ 100 is needed in the occupational exposure risk assessment. However, when a risk assessment for residential uses is conducted in the future, an MOE ≥ 1000 will be needed.

A. Dietary Risk

HED has completed a revision of the dietary risk assessment for methyl parathion using available data and updated methods for estimating acute dietary exposure. Based on the results of the HIARC, hazard endpoints have been selected for both acute (one day) and chronic (long term) exposure intervals. Acute and chronic risk assessments were conducted for all methyl parathion food uses combined, and additional risk assessments were conducted minus individual commodities or commodity subgroups depending on their estimated contribution to the overall dietary exposure. Risk estimates are provided for the average U.S. population and various subgroups, with the major emphasis placed on the exposure estimates for infants and children. This assessment concluded that for the pre-mitigation methyl parathion registered uses, the acute dietary risk estimates exceeded the aPAD for all population subgroups. However, the risk estimates for the post-mitigation remaining uses do not exceed the aPAD for any population subgroup. The assessment also concluded that for pre- and post-mitigation uses, the chronic risk estimates did not exceed the cPAD.

1. Endpoints/Doses for Dietary Risk Assessment

Estimates for one-day, or acute, dietary exposure(s) are compared to an aPAD of 0.00011 mg/kg bw/d, based on a NOAEL of 0.11 mg/kg/d and an uncertainty factor of 1,000. The NOAEL was established in a one-year oral gavage study in rats which demonstrated plasma, RBC, and brain ChE inhibition and neuropathology at 0.53 mg/kg/d. Based on evidence of neuropathology in 3 submitted studies and literature reports (see FQPA Considerations) of sensitivity in young animals (triggering a requirement for a developmental neurotoxicity study), the FQPA safety factor of 10 has been retained and added to the UF of 100 used to account for intraspecies variability and interspecies extrapolation. Acute risk is expressed as a percentage of the aPAD.

Estimates for chronic exposure(s) are compared to a cPAD of 0.00002 mg/kg bw/d, based on a NOAEL of 0.02 mg/kg/d and an uncertainty factor of 1,000. The NOAEL was established in a 2-year rat feeding study which demonstrated RBC ChE inhibition, neuropathology, and systemic toxicity at 0.21 mg/kg/d. Based on evidence of neuropathology in 3 submitted studies and literature reports of sensitivity in young animals, the FQPA safety factor of 10 has been retained and added to the UF of 100 used to account for intraspecies variability and interspecies extrapolation. Risk is expressed as a percentage of the cPAD.

2. Usage Data

Dietary risk estimates were based, in part, on estimates of the percent usage of methyl parathion on each registered food commodity. BEAD estimated methyl parathion use (I. Yusuf and T. Kiely memo, 4/13/99) based on available pesticide survey usage data for the years 1987 through 1997. BEAD estimates were provided to HED as a weighted average, and as a maximum. To be consistent with HED guidance and to avoid underestimating exposure, this risk assessment assumed 1 % crop treated for any BEAD estimate less than 1% (including zero), and also used the estimated maximum percent crop treated (%CT) for each commodity for both the acute and chronic risk assessments. Percent crop treated estimates varied from less than 1% to a maximum of 39% for peaches (Attachments 10 and 11).

3. Residue Data Sources

Methyl parathion residue estimates in this assessment are based primarily on three data sources:

- ① field trial data, submitted by the registrant to support tolerances;
- ② USDA Pesticide Data Program (PDP) food sampling data; and
- ③ Food and Drug Administration (FDA) Surveillance Monitoring data.

The order of preference for the purpose of risk assessment is PDP data > FDA data > field trial data. PDP data are preferred over FDA data because of the statistical design of the PDP program specific for dietary risk assessment, and because the foods are prepared before analysis as they would typically be before consumption (peeling, washing). Methyl parathion commodities not sampled by the PDP program are assessed based on translation of data from PDP sampled commodities in the same crop group, FDA surveillance data, or field trial data. Field trial residue data are generally considered by HED as the upper-end of possible residue more suited to the requirements of tolerance setting than to the requirements of dietary risk assessment (when the most realistic estimate is desired).

When using crop field trial data in this assessment, all data were handled similarly except the data for cottonseed meal. Due to a low pre-harvest interval (PHI) for some special local needs (SLN) on cottonseed grown in Texas, the crop field trial studies were used for cottonseed meal and incorporated Texas %CT only for cotton grown in Texas, as well as the U.S. %CT for cotton grown in all other states, so as not to overestimate the risk (Attachments 10 and 11).

a. Acute exposure

Single Serving Commodities with PDP/FDA Detections: The PDP and FDA databases report detected residues as residues found in 5 lb. composite samples. This manner of reporting may not be representative of possible high-end residues that could be found if individual units of fruits and vegetables were analyzed. This assessment has used a statistical methodology for applying existing (composite) information to acute dietary risk assessments. This methodology consists of extrapolating data on pesticide residues in composite samples of fruits and vegetables to residue

levels in single servings of fruits and vegetables. Given the composite sample mean, the composite sample variance, the number of units in each composite sample, and assuming a log normal distribution, it is possible to *estimate* the mean and variance of the pesticide residues present on single servings of fruits and vegetables. These parameters can then be applied to generate information on the level of residue in fruits and vegetables (and calculate a theoretical distribution). This information can be incorporated into a probabilistic exposure estimation model, such as the Monte Carlo method. This methodology has a higher degree of accuracy when more than 30 composite samples have detectable residues. Commodities that are blended (such as juices) or are smaller than single unit servings (peas) were not decomposited since the measured PDP levels were assumed representative of the actual range of residue.

b. Chronic exposure

For chronic risk assessment, reported residues were averaged, whether based on PDP, FDA, or field trials. If a commodity had no reported detections by the PDP and FDA programs, and the expectation of no detection was confirmed by field trial data, the weighted average of the Limits of Detection (LOD) were used to account for possible exposure that could not be more precisely quantified ($\frac{1}{2}$ LOD methyl parathion + $\frac{1}{2}$ LOD methyl paraoxon).

c. Methyl Paraoxon

This assessment assumes that methyl paraoxon is of equal toxicity as the parent methyl parathion and has accounted for the possibility of this metabolite occurring in treated foods. In general, field trial studies have included analysis for methyl paraoxon, as has FDA surveillance. The PDP program has not analyzed for methyl paraoxon. For the commodities that methyl paraoxon was detected in the field trial data, but not detected by FDA surveillance, paraoxon is accounted for by an assumption of $\frac{1}{2}$ LOD. For commodities with no detection of methyl paraoxon in FDA or field trial data, the assumption was zero residue, and $\frac{1}{2}$ LOD was not incorporated.

d. Processing Factors

Methyl parathion residues may be concentrated, or reduced, by the activities of drying (raisins etc.), processing (juice, catsup etc.), washing, peeling, and cooking. If methyl parathion was measured prior to any of these processes, the predicted effect of the process has been applied to the estimated final residue at consumption. This assessment used factors to account for various processing, but most significantly, for the effect of cooking. This assessment reduced all food-forms designated as boiled, or canned by a factor of 95% (0.05), which was established in a submitted canned snap bean study (MRID 44812901). Other processing factors, including DEEM™ default factors that were used in this assessment are listed in Attachment 12.

4. Consumption Data/DEEM™ Software

The DEEM™ Program: HED is currently using software developed by Novigen Sciences, Inc., named the *Dietary Exposure Evaluation Model*, or DEEM™, to calculate acute and chronic dietary risk estimates for the general U.S. population and various population subgroups. The food consumption data used in the program is taken from the *USDA Continuing Survey of Food Intake by Individuals* (CSFII). The Agency is currently using 1989-92 consumption data. Consumption data are averaged for the entire U.S. population, and within population subgroups such as “all infants” to support chronic risk assessment, but retained as individual consumption data points to support acute risk assessment (which is based on distributions of consumption estimates for either deterministic- or probabilistic-type exposure estimates). The DEEM software is capable of calculating probabilistic (Monte Carlo) type risk assessments when appropriate residue data (distributions of residue) are available.

For acute risk assessments, one-day consumption data are summed and a food consumption distribution is calculated for each population subgroup of interest. The consumption distribution can be multiplied by a residue point estimate for a deterministic (Tier I/II type) exposure/risk assessment, or used with a residue distribution in a probabilistic (Monte Carlo) type risk assessment.

For chronic risk assessments, residue estimates for foods (e.g. apples) or food-forms (apple juice) of interest are multiplied by the averaged consumption estimate of each food/food-form of each population subgroup. Exposure estimates are expressed in mg/kg bw/d and as a percent of the cPAD.

5. Dietary (Food) Risk Results

a. Acute Dietary Risk Before Mitigation Measures

Based on the acute dietary exposure analysis as described above and using an aPAD of 0.00011 mg/kg/d, acute dietary exposure to all population subgroups, pre-mitigation, exceeded the aPAD at the 99.9th exposure percentile. Children 1-6 years were identified as the most highly exposed population subgroup. Estimated acute dietary exposure to children 1-6 years exceeded the aPAD at the 99th and 95th exposure percentiles (See Table 3 following), but did not exceed the aPAD at the 90th exposure percentile. A complete listing of the acute dietary results are in attachment 6.

Several crops were identified as making substantial contributions to the dietary risk. Residues measured on these crops and the surveyed consumption of these crops, factored together, results in these crops taking up a substantial percentage of the “risk cup” and thereby, making substantial contributions to the risk. Theoretically, the risk cup is full when the aggregate risk (food + water + residential) \geq 100% PAD. A number of crops had significant residues from PDP data **and** are high consumption items (e.g. peaches, apples). The acute substantial contributors have been identified as apples, cottonseed, peaches, grapes, and pears. For all the substantial contributors, except cottonseed oil, PDP and/or FDA monitoring data have shown measurable residues of methyl parathion, some greater than half the tolerance. The FDA monitoring data used for cottonseed oil showed no detectable residues; however, there were only two samples of oil analyzed. The Agency believes that residues are not likely to be found in cottonseed oil since there are no detectable residues found in cottonseed. Therefore, FDA monitoring data were used so as not to overestimate the potential risk from cottonseed oil.

The acute summary table below shows the acute dietary risks to the U.S. population, infants, and children from exposures to all the supported crops, pre-mitigation (See Attachment 6).

Table 3. Pre-mitigation Acute Dietary Risk Estimates

Population	(95th percentile)		(99th percentile)		(99.9th percentile)	
	Exposure	% aPAD	Exposure	% aPAD	Exposure	% aPAD
U.S. Population	0.000044 mg/kg/day	40	0.000121 mg/kg/day	110	0.000416 mg/kg/day	378
All Infants <1 year	0.000095 mg/kg/day	86	0.000169 mg/kg/day	153	0.000415 mg/kg/day	377
Children 1-6 years	0.000132 mg/kg/day	120	0.000273 mg/kg/day	249	0.000969 mg/kg/day	881
Children 7-12 years	0.000061 mg/kg/day	55	0.000129 mg/kg/day	117	0.000428 mg/kg/day	388

b. Chronic Dietary Risk Before Mitigation Measures

Based on the chronic pre-mitigation dietary exposure analysis as described above and using an cPAD of 0.00002 mg/kg/d, chronic dietary exposure to all population subgroups did **not** exceed the cPAD (See Table 4 following). Children 1-6 years were identified as the most highly exposed population subgroup. The chronic summary table below shows the chronic dietary risks to the U.S. population, infants, and children from exposures to all the supported crops, pre-mitigation, for which methyl parathion is registered (Attachment 7). The chronic substantial contributors have been identified as apples, peaches, grapes, cottonseed oil, and pears. For all the substantial contributors, except cottonseed oil, PDP and/or FDA monitoring data have shown measurable residues of methyl parathion. The FDA monitoring data used for cottonseed oil showed no detectable residues; however, there were only two samples of oil analyzed.; however, there are not sufficient USDA/FDA monitoring data reported for residues of methyl parathion in/on cottonseed oil. Since monitoring data showed significant residues on cottonseed meal (feed use), the Agency believed it likely that residues could be found in cottonseed oil. Therefore, field trial data were used so as not to underestimate the potential risk. The crop field trial studies were used for cottonseed oil incorporating Texas %CT for cotton grown in TX, and U.S. %CT for cotton grown in all other states (Attachment 7).

Table 4. Pre-mitigation Chronic Dietary Risk Estimates

Population	Exposure (mg/kg/day)	% Chronic PAD
U.S. Population	0.000003	17
All Infants (<1 year)	0.000006	29
Children 1-6 years	0.000009	47
Children 7-12 years	0.000005	22

6. Dietary Risk Reflecting Mitigation Measures

a. Recent Use Changes - Remaining Uses

The uses for methyl parathion reflecting mitigation measures include almonds, barley, dried beans, cabbage, canola oil (rape seed oil), field corn, sweet corn, cottonseed, lentils, oats, onions, peanuts, dried peas, pecans, potatoes, rice, rye, soybeans, sugar beets, sunflowers, sweet potatoes, walnuts, and wheat.

b. Acute Dietary Risk Reflecting Mitigation Measures

Based on the acute dietary exposure analysis as described above and using an aPAD of 0.00011 mg/kg/d, acute dietary exposure to all population subgroups, acute dietary risks reflecting mitigation measures, do not exceed the aPAD at the 99.9th exposure percentile (Table 5). A complete listing of the acute dietary risk calculation results are in attachment 8.

Table 5. Post-mitigation Acute Dietary Risk Estimates

Population	(95th percentile)		(99th percentile)		(99.9th percentile)	
	Exposure	% aPAD	Exposure	% aPAD	Exposure	% aPAD
U.S. Population	0.000027 mg/kg/day	24	0.000042 mg/kg/day	38	0.000068 mg/kg/day	60
All Infants <1 year	0.000033 mg/kg/day	30	0.000051 mg/kg/day	47	0.000067 mg/kg/day	61
Children 1-6 years	0.000043 mg/kg/day	39	0.000056 mg/kg/day	50	0.000086 mg/kg/day	78
Children 7-12 years	0.000032 mg/kg/day	29	0.000042 mg/kg/day	38	0.000087 mg/kg/day	78

c. Chronic Dietary Risk Reflecting Mitigation Measures

Based on the chronic dietary exposure analysis reflecting mitigation measures and using a cPAD of 0.00002 mg/kg/d, chronic dietary risk to all population subgroups does **not** exceed the cPAD (See Table 6 following). A complete listing of the chronic dietary risk calculation results are in attachment 9.

Table 6. Post-mitigation Chronic Dietary Risk Estimates

Population	Exposure (mg/kg/day)	% Chronic PAD
U.S. Population	0.000001	3
All Infants (<1 year)	0.000001	3
Children 1-6 years	0.000002	8
Children 7-12 years	0.000001	5

7. Conclusions

Apples, peaches, grapes, and pears were found to be substantial contributors to both the acute and chronic dietary risk based on PDP detects and high consumption. Decomposited PDP data were used for the residue distribution file for peach residues. The decomposited data were truncated to eliminate the highest 25 residues and no substantial effect on the exposure was found indicating that the high residues obtained as a result of decomposing are minimally reflected in the overall exposure.

The mitigation measures **remove many of the substantial contributors**, particularly commodities consumed by children, and greatly lower the potential dietary exposures to both adults and children.

B. Drinking Water Risk

1. Acute Drinking Water Risk Per Pre-mitigation Measures

Generally, the Agency calculates Drinking Water Levels of Comparison (DWLOC) for comparison to measured or modeled drinking water concentrations for the risk analysis. The DWLOC is the concentration in drinking water, as part of the aggregate exposure, that occupies no more than 100% of the PAD. The dietary exposure and DWLOC together, cannot be greater than 100% of the PAD. Any measured or modeled drinking water estimates that are less than the DWLOC are not of concern.

Acute exposures from methyl parathion in drinking water may add to the dietary risk. The DWLOC for acute exposure was calculated to be zero since the acute exposure from food alone on all registered use sites is > 100% of the aPAD.

2. Chronic Drinking Water Risk from Surface Water Per Pre-mitigation Measures

Non-targeted surface water survey studies performed over the past 30 years have not shown concentrations of methyl parathion at levels predicted in the chronic modeling assessments (4.2 ppb). The average reported value from the Louisiana composites of intake water is 0.009 ppb. A chronic DWLOC (DWLOC_{chronic}) was calculated using the following formulae:

$$\text{DWLOC}_{\text{chronic}} (\mu\text{g/L}) = \frac{\text{chronic water exposure (mg/kg/d)} \times \text{body weight (kg)}}{\text{consumption (L/d)} \times 10^{-3} \text{ mg}/\mu\text{g}}$$

$$\text{chronic water exposure (mg/kg/d)} = [\text{cPAD} - \text{chronic food (mg/kg/d)}]$$

The current Agency default body weight and consumption values are 10 kg and 1 liter/day, respectively, for all infants and children, 70 kg and 2 liters/day for adult males, and 60 kg and 2 liters/day for adult females. These default values and others are presently under review in the Agency. If at a future time the Agency decides to change the default assumptions used, the impact of the changes on the methyl parathion risk assessment will be considered.

Table 7. Chronic Surface Water

Population	Monitoring Data (ug/L)	cPAD (mg/kg/d)	Chronic Food Exposure (mg/kg/d)	Chronic H ₂ O Exposure (mg/kg/d)	DWLOC _{chronic} (ug/L)
Adult Male	0.009	0.00002	0.000002	0.000018	0.63
Adult Female	0.009	0.00002	0.000005	0.000015	0.45
Infants <1 yr	0.009	0.00002	0.000006	0.000014	0.14
Children 1-6	0.009	0.00002	0.000009	0.000011	0.11

Concentrations from available monitoring studies were well below the OW's 2 ppb HA. Although the available chronic monitoring data do not allow a comprehensive assessment, EFED believes that chronic concentrations of methyl parathion in surface water will be below the 2 ppb HA. The table above shows the limited monitoring concentration of 0.009 ppb does not exceed the DWLOC_{chronic}. As mentioned earlier, these data do not represent concentrations after drinking water treatment and may actually be lower.

3. Chronic Drinking Water Risk from Ground Water Per Pre-mitigation Measures

It is uncertain whether chronic exposures from ground water would pose a risk concern without any targeted monitoring studies. No model exists for specifically estimating chronic ground water concentrations. Therefore, a highly conservative modeled ground water concentration of 0.6 ppb (from the acute model) is the default concentration. However, EFED believes it is very unlikely that chronic exposures would be as high as 0.6 ppb. The DWLOCs_{chronic} are the same as for surface water concentrations.

4. Drinking Water Risks Reflecting Use Mitigation Measures

Based on use changes reflecting mitigation measures, the acute and chronic exposures to methyl parathion in food have been reduced. The Agency recalculated the DWLOCs for chronic risk analysis to reflect these changes. Since the acute exposures from food no longer exceed the aPAD, DWLOCs_{acute} were also calculated.

Surface water monitoring data range between 6 ppb from methyl parathion applications to rice fields in California to 0.42 ppb from applications to cotton in Mississippi. After the monitoring data were recorded in California, the state instituted a number of its own mitigation

measures to reduce contamination of surface waters and therefore, present-day concentrations would be expected to be lower. As a result, EFED has more confidence in the surface water concentrations from Mississippi (0.42 ppb) and it should be noted that cotton has the highest application rate for methyl parathion than any other remaining uses.

Table 8. Acute Surface Water Reflecting Use Mitigation Measures

Population	Monitoring Data (ug/L)	aPAD (mg/kg/d)	Acute Food Exposure (mg/kg/d)	Acute H ₂ O Exposure (mg/kg/d)	DWLOC _{acute} (ug/L)
Adult Male	0.42	0.00011	0.000067	0.000043	1.51
Adult Female	0.42	0.00011	0.000075	0.000035	1.05
Infants <1 yr	0.42	0.00011	0.000067	0.000043	0.43
Children 1-6	0.42	0.00011	0.000087	0.000023	0.23

Though comparisons between the untreated surface water monitoring data and the DWLOC_{acute} for children 1-6 years of age raise some concerns, it is uncertain what the actual “at the tap” drinking water residues would be after dilution from the source to the tap and after treatment. Since these Mississippi monitoring data come from a high use region (cotton has the highest application rate), the Agency believes that they are somewhat conservative though recognizably limited.

Table 9. Chronic Surface Water Reflecting Use Mitigation Measures

Population	Monitoring Data (ug/L)	aPAD (mg/kg/d)	Acute Food Exposure (mg/kg/d)	Acute H ₂ O Exposure (mg/kg/d)	DWLOC _{chronic} (ug/L)
Adult Male	0.009	0.00002	0.000001	0.000019	0.67
Adult Female	0.009	0.00002	0.000001	0.000019	0.57
Infants <1 yr	0.009	0.00002	0.000001	0.000019	0.19
Children 1-6	0.009	0.00002	0.000002	0.000018	0.18

Based on the limited chronic drinking water data, potential residues of methyl parathion in water are not of concern. The chronic monitoring data were collected closer to the tap (drinking water intake) over a period of a year from a high use area and therefore, are approaching what may be actual residues in “at the tap” drinking water.

It is uncertain whether exposures from ground water would pose a

risk concern without any targeted monitoring studies. The highly conservative modeled ground water concentration of 0.6 ppb from the acute model is the estimated concentration for both the acute and chronic ground water drinking water estimates. However, EFED believes it is very unlikely that any ground water exposures would be as high as 0.6 ppb, based on fate information. The DWLOCs_{acute} and DWLOCs_{chronic} are the same as for surface water concentrations.

5. Considerations

There are several things to consider when weighing the potential contribution to the total dietary risk from drinking water contaminated with methyl parathion. The limited monitoring data available to the Agency indicate that exposures would be expected to be lower than the modeled estimates. In addition, neither the models nor the monitoring data reflect concentrations after dilution (from source to treatment to tap) or drinking water treatment. Methyl parathion is a compound that can be absorbed onto activated carbon as a water treatment method. However, Granulated Activated Carbon (GAC) is not a commonly used technology, and it is expensive to install and maintain. Less than 1% of the 55,000 community water treatment systems in the United States use GAC filters. A community water treatment system is defined as serving more than 25 people or having 15 or more service connections. GAC is most often used to remove pesticides, to control odor, and taste problems. There are currently little data on the efficacy of other more common treatment technologies in removing methyl parathion.

When the available monitoring data were gathered, methyl parathion was measured, but they did not look for methyl paraoxon. EFED does not have any data available with which to predict the rate of formation, or the half-life of, methyl paraoxon. Though there are data to show that another organophosphate, malathion, degrades to its oxon metabolite during drinking water treatment, it is unknown if methyl parathion would behave in a similar manner.

Given the fact that the monitoring data represent only a very small range of conditions (regional weather, streamflow, application rates and methods), it cannot be assumed that they represent surface water concentrations or conditions elsewhere in the United States, and the Agency still does not have any ground water monitoring data. The data collected closest to the tap (treatment plant intake) in Louisiana do not indicate exposures that would be of concern. Though the Agency considers it unlikely that drinking water concentrations “at the tap,” will

make the largest, or a significant, contribution to the total dietary burden, there is sufficient information from available monitoring data and models to warrant close monitoring of potential surface and ground water sources of methyl parathion exposure.

C. Occupational/Residential Risk

1. Combined Dermal and Inhalation Risk from Handler Exposures

While the MOEs for the pre mitigation uses and the post mitigation uses vary, the scenarios that pose a risk of concern are the same for both. Dermal and inhalation exposures were combined and risk was calculated for each exposure scenario using the short- and intermediate-term dermal and inhalation NOAEL of 0.11 mg/kg/day and 100% dermal absorption and inhalation absorption. An $MOE \geq 100$ is needed for the risk to be acceptable. Overall, there is moderate to high confidence in the PHED data from which the occupational exposures used in the assessment were derived. See Tables 2-4 in Attachment 14 for details. The calculations of risk based on combined dermal and inhalation exposure indicate that the MOEs are less than 100 even with maximum risk reduction measures (inside the cab of a truck) for all of the short and intermediate term scenarios listed except for the flagging at the lowest application rates.

- ☐ Flagging aerial spray applications with engineering controls for the EC formulation at the 0.375 lbs ai/A application rate (MOE = 260).
- ☐ Flagging aerial spray applications with engineering controls for the Mcap formulation at the 0.5 lbs ai/A application rate (MOE = 190).

One of the registrants has stated that they are not supporting the use of human flaggers. However, HED has included the risk to flaggers in this assessment because some current labels allow the use of flaggers.

2. Postapplication Risk

a. Microencapsulated Formulation

The surrogate postapplication assessment for pre mitigation uses indicates that following applications of methyl parathion to grapes at 1.5 lbs ai/A and 3.0 lbs ai/A workers cannot reenter the fields for 30 and 33 days, respectively, without being exposed to levels of methyl parathion that would result in MOEs of less than

100:

- ❑ MOEs ≥ 100 for grapes at the registrant suggested application rate of 1.5 lbs ai/A with a dermal transfer of 15,000 cm²/hr at the **30th day following application.**

- ❑ MOEs \geq 100 for grapes at the current label application rate of 3.0 lbs ai/A with a dermal transfer of 15,000 cm²/hr at the **33rd day following application**.
- ❑ MOEs \geq 100 for tree crops such as pears, apples, and peaches with a dermal transfer of 10,000 cm²/hr at the **30th day following application** (2.0 lbs ai/A).

The surrogate postapplication assessment for the post mitigation uses indicates that:

- ❑ MOEs \geq 100 for nut crops including pecans, almonds and walnuts with a dermal transfer of 10,000 cm²/hr at the **30th day following application**.

b. Emulsifiable Concentrate Formulation

The post application assessment for the emulsifiable concentrate formulation is the same for the pre mitigation uses and the post mitigation uses. The surrogate postapplication assessment indicates that:

- ❑ MOEs \geq 100 for cotton - early season, with a dermal transfer of 1,000 cm²/hr on the **7th day after application** (3.0 lbs ai/A).
- ❑ MOEs \geq 100 for cotton - late season, with a dermal transfer of 4,000 cm²/hr on the **9th day after application** (3.0 lbs ai/A).

3. Residential Risk

Risk estimates of residential dermal and inhalation exposures were not estimated. The Agency is currently developing methods to assess residential risks, and these risks will be assessed in the future when these new methods are available. However, based on available information, HED remains concerned about residential risks from methyl parathion spray drift.

D. Aggregate Risk

Under the Food Quality Protection Act, the Agency considers contributions to risk from various exposure sources, specifically, food, drinking water, and residential. Methyl parathion has no registered residential uses, therefore only exposures through food and drinking water were considered in the aggregate risk assessment.

The potential for other non-occupational exposures to individuals living in or near agricultural areas where methyl parathion is being used were not included in the aggregate risk assessment but will be addressed at a later time when methodologies to perform such assessments are in place.

The acute aggregate risk estimate for all registered uses, pre-mitigation, indicated that there is no room for exposure to methyl parathion in drinking water because risk from food sources alone exceed the Agency's level of concern (i.e. > 100% acute PAD). The acute aggregate risk estimate which reflects mitigation measures may still be of concern. Though acute exposure to methyl parathion from food sources alone, with mitigation measures, does not exceed the Agency's level of concern (i.e. < 100% acute PAD), limited surface water monitoring data indicate potential exposures at unacceptable levels. However, as discussed earlier, the monitoring data are not nationally representative, do not represent dilution from the source to the tap, and do not reflect water treatment. Without actual drinking water monitoring data, it is difficult to draw any conclusions about actual residues in drinking water.

The chronic aggregate risk assessment is not of concern, pre- or post-mitigation measures. In particular, chronic exposures reflecting mitigation measures to methyl parathion from food sources alone are well below the Agency's level of concern (i.e. < 100% chronic PAD). Limited drinking water monitoring data indicate drinking water exposures may be very low. In addition, fate data show that methyl parathion is not persistent.

E. Cumulative Risk

The Agency is in the process of formulating guidance for conducting cumulative risk assessment. When the guidance is completed, peer reviewed, and finalized, methyl parathion and other organophosphates will be revisited to assess the cumulative effects of exposure to multiple organophosphates.

IV. Data Needs

A. Developmental Neurotoxicity Study

A Developmental Neurotoxicity Study is required.

B. Tolerance Reassessment Data

Data needs for the tolerance reassessment and dietary risk assessment are summarized as follows:

1. Plant and Animal Metabolism Data

Pending acceptance of recently submitted lettuce metabolism data, and additional goat and hen metabolism data, which are under review, no additional plant and animal metabolism data will be required to support the reregistration of methyl parathion. The registrant should resubmit the goat and hen metabolism data cited above through the MRID process.

The Agency continues to recommend that future plant and animal magnitude of the residue studies include data depicting residues of *p*-nitrophenol resulting from the use of methyl parathion.

2. Analytical Methods - Plant and Animal

Since the proposed enforcement method(s) is/are the FDA multiresidue testing protocol(s), an independent laboratory validation (ILV) is not required.

In conjunction with the ruminant and poultry feeding studies, the registrants must provide data validating the analytical method(s) used for determining methyl parathion, methyl paraoxon, *p*-nitrophenol, and amino-paraoxon-methyl in meat, milk, poultry, and eggs. If the feeding studies indicate that tolerances are necessary for residues in animal commodities, then the registrants must propose an enforcement method for determining the residues of concern in animal commodities which must be regulated.

3. Storage Stability Data

HED acknowledges receipt of the new storage stability data on plants submitted in support of the reregistration of methyl parathion (Attachment 4). These data are under review and pending acceptance of these new data to satisfy guideline requirements, no additional storage stability data on plant commodities will be required to support the reregistration of methyl parathion.

Data depicting the storage stability of methyl parathion residues of concern in animal commodities are required in conjunction with the ruminant and poultry feeding.

4. Magnitude of the Residue Data - Plant Commodities

HED acknowledges receipt of new residue chemistry data submitted in support of the reregistration of methyl parathion (Attachment 4), which are under review and which have been used in the residue chemistry science assessments and dietary risk assessment analyzes for methyl parathion as the Agency deems appropriate.

HED understands that Cheminova has committed to generate alfalfa field trial data (Received 05/99), grass field trial data (Received 05/99), cotton gin by-product magnitude of the residue data, and sunflower seed processing data in support of the registration of the EC formulation of methyl parathion.

HED understands that Elf Atochem has committed to generate potato field trial data, onion field trial data, soybean field trial data, plum field trial data, cotton gin by-product magnitude of the residue data, and plum processing data in support of the reregistration of the Mcap formulation of methyl parathion. Potato data will be translated to support the use of the Mcap formulation of methyl parathion on sweet potatoes and yams.

Additional residue chemistry data are required to support the reregistration of methyl parathion which the registrants (Cheminova and Elf Atochem) have not committed to generate. Additional sugar beet top, turnip top, wheat forage, and wheat hay magnitude of the residue data are required to support the reregistration of the EC formulation of methyl parathion. Additional pear field trial data and rice straw magnitude of the residue data are required to support the reregistration of the Mcap formulation of methyl parathion.

5. Magnitude of the Residue Data - Animal Commodities

Reregistration requirements for magnitude of the residue in meat, milk, poultry, and eggs remain outstanding. No tolerances have been established for residues of methyl parathion in animal commodities, although tolerances have been established on numerous animal feed items. HED understands that the registrants have committed to generate these data.

C. Occupational Handler Exposure/Risk Data

The occupational handler risks for all but one exposure scenario are of concern. Specific exposure studies and data needs will be addressed after risk/risk mitigation concerns are addressed.

D. Occupational Post-application Exposure/Risk Data

The occupational post-application risks for the EC and Mcap formulations are of concern. Specific post-application exposure studies and data needs will be addressed after risk/risk mitigation concerns are addressed.

E. Dioxin Data

Product analyzes for dioxins at LOQ = 0.1 ppb as requested by 6/87 DCI.

V. List of Attachments

- 1 - Methyl Parathion (O,O-dimethyl O-p-nitrophenyl phosphorothioate): Hazard Identification Committee Report (George Ghali, December 1, 1997)
- 2 - Methyl Parathion - Re-evaluation of Dietary Endpoint and Non-dietary Endpoint Selection and Dermal Absorption Factor; Report of the Hazard Identification Assessment Review Committee (Kathleen Raffaele, March 23, 1999)
- 3 - Revised Toxicology Chapter (Kathleen Raffaele, 06/01/99)
- 4 - Revised Residue Chemistry Chapter for the Methyl Parathion Reregistration Eligibility Decision (RED) Document (Bonnie Cropp-Kohlligian, 05/12/99)
- 5 - Methyl Parathion (053501). The Outcome of the HED Metabolism Assessment Review Committee Meeting Held on March 11, 1998 (Bonnie Cropp-Kohlligian, May 21, 1998)
- 6 - Pre-mitigation Acute Dietary Monte Carlo Assessment
- 7 - Pre-mitigation Chronic Dietary Assessment
- 8 - Post-mitigation Acute Dietary Monte Carlo Assessment
- 9- Post-mitigation Chronic Dietary Assessment
- 10- Raw Data Table
- 11- Anticipated Residue Determination for Acute Dietary Assessment
- 12 - DEEM memo

- 13 - Residue Data Files
- 14 - Revised Occupational and Residential Exposure Assessment and Recommendations for the Reregistration Eligibility Decision Document for Methyl Parathion (Jonathan Becker and Renee Sandvig, 07/30/99)
- 15 - Review of Methyl Parathion Incident Reports (Jerome Blondell and Monica Spann, February 5, 1998)
- 16 - Revised Product Chemistry Chapter for the Methyl Parathion Reregistration Eligibility Decision (RED) Document (Ken Dockter, 05/25/99).